

The University of Chicago

FACTORS CONTROLLING BRAIN
POTENTIALS IN THE CAT

A DISSERTATION SUBMITTED TO THE FACULTY
OF THE DIVISION OF THE BIOLOGICAL SCIENCES
IN CANDIDACY FOR THE DEGREE OF DOCTOR OF
PHILOSOPHY

DEPARTMENT OF PHYSIOLOGY

1939

By

HAROLD H. DUBNER

Private Edition, Distributed by
THE UNIVERSITY OF CHICAGO LIBRARIES
CHICAGO, ILLINOIS

Reprinted from
THE JOURNAL OF NEUROPHYSIOLOGY
Vol. 2, No. 2, March 1939

The University of Chicago

FACTORS CONTROLLING BRAIN POTENTIALS IN THE CAT

A DISSERTATION SUBMITTED TO THE FACULTY
OF THE DIVISION OF THE BIOLOGICAL SCIENCES
IN CANDIDACY FOR THE DEGREE OF DOCTOR OF
PHILOSOPHY

DEPARTMENT OF PHYSIOLOGY

1939

By
HAROLD H. DUBNER

Private Edition, Distributed by
THE UNIVERSITY OF CHICAGO LIBRARIES
CHICAGO, ILLINOIS

Reprinted from
THE JOURNAL OF NEUROPHYSIOLOGY
Vol. 2, No. 2, March 1939

FACTORS CONTROLLING BRAIN POTENTIALS IN THE CAT*



HAROLD H. DUBNER

From the Department of Physiology, University of Chicago

(Received for publication January 3, 1939)

IT IS NOW ESTABLISHED that central neurones may exhibit spontaneous rhythmic potentials. Even when synaptic transmission is blocked these spontaneous waves continue and may increase in amplitude (Libet and Gerard, 1938). We have studied in the cat some of the physical and chemical factors that influence this "intrinsic" periodicity, as well as the effect on it of external stimulation under varying physico-chemical conditions; certain neural factors affecting the rhythm have also been investigated.

METHOD

The left hemisphere of cats was widely exposed under light nembutal anaesthesia (35 mg. per kg. intraperitoneally), and the Horsley-Clarke instrument attached. An hour later a concentric needle was placed in the desired position along the optic pathways and amplified potentials recorded with a crystallograph or cathode ray oscillograph. Retinal stimulation was caused usually by a flash-light directed into the contralateral eye, or sometimes by a single flash of a condenser discharge through a 2.5 V. bulb. A polarizing current (less than 1.0 mA.) was passed through brain tissue between an indifferent silver plate (2 to 3 cm. surface) applied to the surface of the opposite cerebral cortex, and a silver wire (2 to 3 mm. uninsulated tip) placed 3 to 4 mm. lateral to the recording electrode. Solutions injected into the carotid artery or femoral vein were isotonic, neutral, and at body temperature; and those applied to the cortex directly were often buffered with phosphate (this did not alter their action, although it increased control potentials). The various procedures were carried out in irregular order over a period of 10 to 12 hr. Additional anesthetic was sometimes needed towards the end of the experiment. Each agent was tested in not less than 4, usually in 7 to 12 experiments.

RESULTS

Neural factors

Evoked and spontaneous potentials (normal). The responses evoked by directed retinal stimulation (contralateral eye) vary in form and amplitude with the position of the leading-off electrodes, intensity of stimulus, and extent of previous "dark-adaptation." There is also a cyclic fluctuation in threshold, especially after repetitive stimulation (Bishop, 1933). Threshold, latency, and form have been studied by Bartley (1934), Wang (1934), and by Gerard, Marshall, and Saul (1936), and the present description is intended as a norm with which to compare changes. The geniculate "on" response consists of an initial negative potential (50 to 90 μ V.) followed by a prolonged positive wave (Fig. 1B, 2 and 3A). Superimposed upon the latter are often additional positive waves which increase with heightened general excitation (Fig. 4A). The "off" is similar in form and direction, but 20 to 50 μ V. less in amplitude. Repeated illuminations evoke in the geniculate, as

* Preliminary reports of this work have appeared in: *Cold Spr. Harb. Mongr.*, 1936, 4: 292; *Trans. Amer. neurol. Ass.*, 1936, 62: 55-60, and *Amer. J. Physiol.*, 1938, 123: 56-57.

in the radiations and cortex (Bartley, 1936), responses which show a semi-rhythmic fluctuation in amplitude and form. "Off" potentials tend to increase on repetition, and the "on" spike may give way to several positive waves. When spontaneous potentials are marked, the responses to light are not discernible, although 1 sec. later they may be strong (Fig. 1Ac).

Throughout the optic pathways a regular rhythm of from 2 to 4 per sec. is present, which usually becomes asynchronous during retinal stimula-

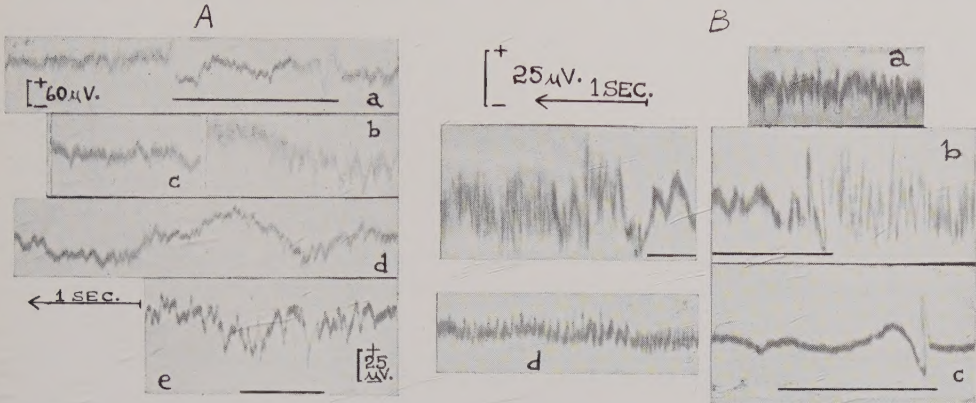


FIG. 1A. Spontaneous and evoked potentials from the lateral geniculate body. After two hours in dark: (a) Response to illumination (black line). Note the initial increase and later suppression of the rapid waves present before illumination; (b) 5 sec. later, note the rhythm at 4.2 a sec.; (c) 10 sec. after (b), rhythm now 2.7 a sec.; (d) moderate diffuse constant illumination, note alternate periods with and without rapid waves; (e) electrode 3 mm. higher, usual "on" and "off" optic response.

FIG. 1B. Lateral geniculate potentials following brain section: (a) Following ipsilateral decortication; (b) 5 min. after subsequent midbrain transection, note greatly increased fast and slow waves, much reduced during illumination; (c) 4 min. after additional nembutal, optic stimulus; (d) 1 hr. after (c).

tion (Gerard, Marshall and Saul, 1936). This rhythm is most prominent with optimum afferent excitation, and was best studied by continuous observations for 6 to 10 hr. at a given locus. After 1 to 2 hr. of absolute darkness, the 2 to 4 per sec. rhythm was often replaced by continuous, irregular, fairly rapid waves. A directed light then gave "on" and "off" responses and inhibited the high frequency, but it did not initiate a rhythm. In diffuse light of moderate intensity (ceiling light on), however, the rhythm reappeared in 4 to 6 sec. This rhythm could be abolished by retinal stimulation with focussed light. The slow rhythm has superimposed on it waves at 8, 20 to 25, and 60 to 80 per sec., all of which are inhibited during light. The 8 and 20 per sec. waves are present only in the lateral geniculate and radiations, and are normally small, 15 and 5 μ V. respectively. They are considerably augmented by increased blood potassium, by strychnine and polarization, and during the after-discharge from retinal stimulation. The 60 per sec. waves of 5 to 10 μ V. are most prominent in the large ventral cells of the lateral geniculate and fade off in the optic tract and radiations. They persist

after ipsilateral decortication and are markedly augmented following mid-brain transection (Fig. 1B), though still eliminated by light directed especially into the contralateral eye. Only under optimal conditions are the slow and the high frequency rhythms distinct in the distal radiations and cortex.

Following a retinal stimulus and the "off" response, the after-discharge modifies for some time the continuous activity. This effect varies with the previous degree of dark adaptation and the number of preceding flashes of light. After a single flash the 3 and 60 per sec. geniculate rhythms return within 5 sec., but following a second flash a high frequency after-discharge develops which lasts 15 to 20 sec. When the slow rhythm returns, it may be half again as fast as normal, e.g., 4.2 per sec. 5 sec. after "off," and 2.7 after another 10 sec. Repeated stimuli (under light anesthesia) induce a regular alternation, at about 1 sec. intervals, with the 60 per sec. rhythm now absent, now present, on the continuing slow waves. This alternation lasts longer after many flashes than after few, but it finally returns to the initial resting state with both rhythms present. Under deeper narcosis, a similar alternation may appear during continuous diffuse illumination of moderate intensity (Fig. 1A a to d), to be inhibited by light directed into the eyes. In the optic tracts, in contrast to the geniculate, the slow rhythm returns almost immediately even after repeated light flashes.

Potentials following section of the brain. Bishop (1933, 1936) attributed the slow rhythm of the optic pathways to reverberating thalamo-cortico-thalamic circuits (Lorente de Nó, 1934, 1935). Interruption of these pathways by uni- or by bilateral removal of the parietal and occipital cortex does not abolish the geniculate rhythm in the cat (Fig. 1Ba). Complete mid-brain transection (Fig. 1Bb), caudal to the colliculi, regularizes the slow rhythm and doubles its amplitude, to 40 μ V., and strikingly augments the 25 per sec. waves, to 25 μ V. These modified rhythms persist for hours; are completely inhibited by light directed into the eyes, and return following it; and they are depressed to less than one-fourth their amplitude within 1 min. of the injection of additional nembutal (15 mg. per kg.), gradually to regain full size as the anesthetic wears off.

Physical factors

Brain polarization. A feeble constant current passed through the brain and concentrated toward a "different" needle electrode, placed just lateral to the pick-up electrode, enormously increases the amplitude of potentials in the optic pathways, especially the lateral geniculate, usually with no change in rate (Fig. 2). The direction of the polarizing current is immaterial, although perhaps the different electrode as anode favored the appearance of waves of high frequency. The augmentation may persist for 10 min. following a 30 sec. polarization. Besides the great increase in the slow geniculate rhythm, the "on" and "off" responses to light are also intensified and sometimes are followed by high frequency discharges. The possibility that these changes are an artefact due to the polarizing current is excluded by

the following observations: the induced increase in the rhythm depends on the brain structure and is very marked only in the geniculate, the effects long outlast the polarization, and at a given locus polarization may slightly or greatly increase the rhythm, depending on the state of the animal and other physiological variables.

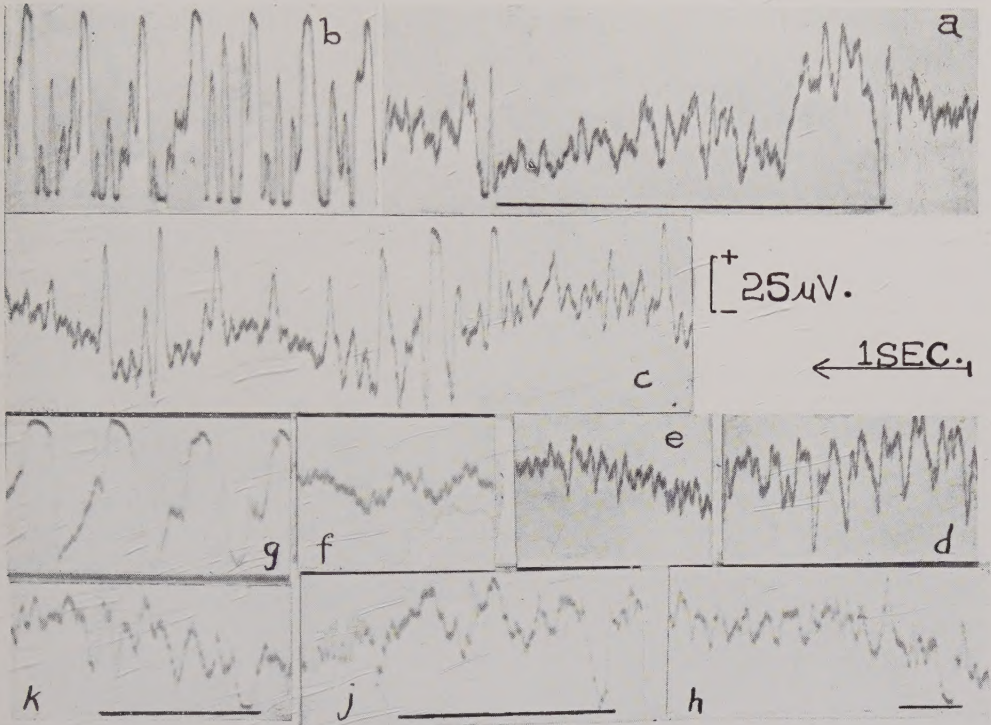


FIG. 2. Potentials modified by brain polarization. Lateral geniculate: (a) Control with light response; (b) during polarization, amplifier overloaded; (c) 10 sec. after polarization; (d) and (e) 2.5 and 13 min. after polarization, respectively. Another cat: (f) control; (g) during polarization. Electrode to optic radiations; (h) control and optic response; (j) during polarization, note rapid waves at "off"; and (k) 10 sec. after polarization.

Chemical factors

Potassium ion. Intravascular injection of isotonic KCl, sufficient to increase the blood potassium from 20 to 50 per cent above normal, induced psychomotor excitation with: gasping respiration, increased deep reflexes, occasional bilateral running movements, urination, loud vocalization, and purposeful scratching gestures. Within 2 min., the slow geniculate rhythm and, especially, the superposed 8 per sec. waves were augmented. When these had again diminished, a retinal stimulus still increased them; 5 min. after injection a continued retinal stimulus gave a somewhat increased "on" response followed by regular 45 per sec. potentials which waxed and waned at 2 per sec. (the frequency of the usual slow rhythm; Fig. 3A). Potentials

of the optic cortex were not clearly affected. Within 10 min. the animal returned to surgical narcosis and the thalamic potentials resumed their pre-injection character. These time relations parallel those of rise in blood po-

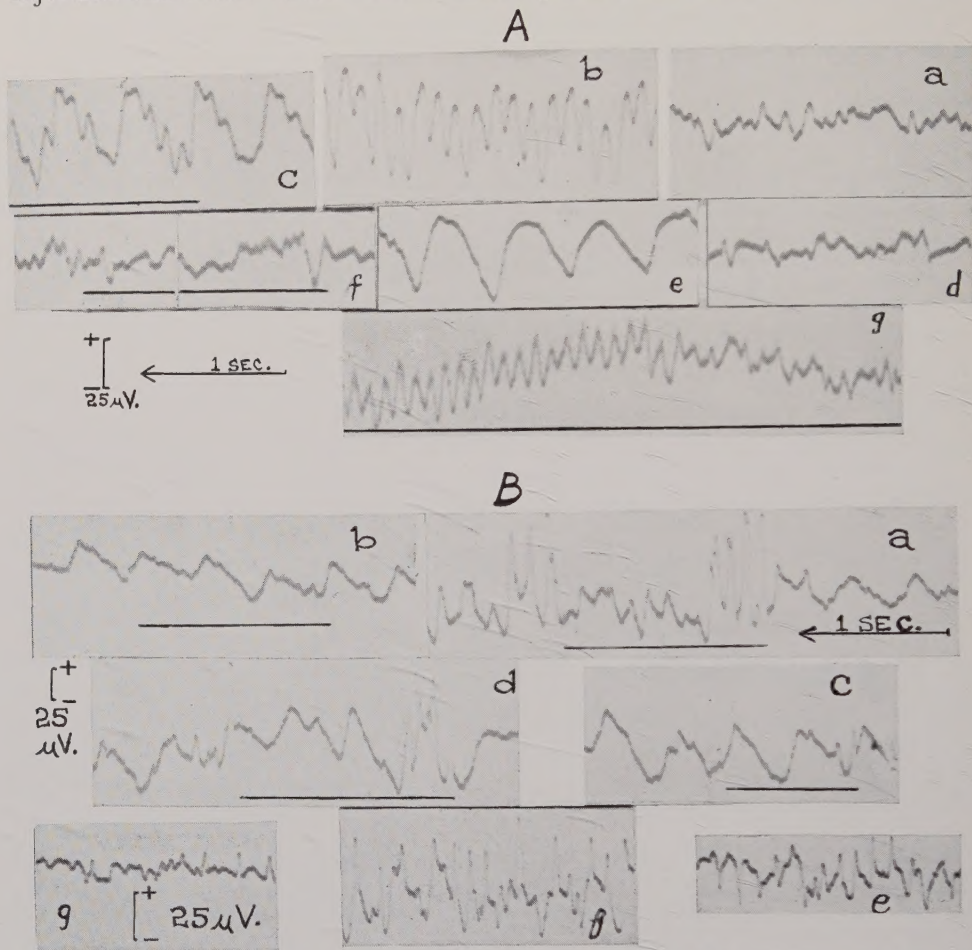


FIG. 3A. Lateralgeniculate potentials as modified by ions and strychnine on intravascular injection. (a) Ringer's solution injected as control; (b) 2 min. after K; (c) 30 sec. after (b); (d) 30 min. later; (e) 2 min. after Ca; (f) 15 min. after (e); (g) 4 min. after strychnine.

FIG. 3B. Striate cortex potentials (surface record) as modified by ions locally: (a) Normal rhythm and optic response; (b) 10 min. after K; (c) 10 min. after (b) and washing with Ringer's solution, note limited recovery; (d) 1 min. after (c) and after washing with Ca. Another animal: (e) 10 min. after K; (f) 15 min. after (e) and after washing with Ringer's solution; (g) 2 min. after Ca.

tassium following intravenous potassium administration (Houssay and Marenzi, 1937). The largest single intravenous dose of KCl tolerated was 15 mg. per kg. in cats; Houssay and Marenzi found 20 mg. per kg. in dogs. Death was apparently due to cardiac failure.

Local application of excess K ion to the striate cortex reversibly depresses and may completely abolish spontaneous and evoked potentials. (With sufficiently weak potassium solutions, the expected increase of potentials is obtained.) Ringer's solution with tripled K ion cuts the amplitude to half in 2 min. with spontaneous recovery in 7 to 10 min. Isotonic KCl completely abolishes the normal "on" and "off" potentials (initially 100 μ V.) by 2 to 10 min. after application (Fig. 3Ba and b). There is little return in another 10 min. even after washing with warm Ringer's solution (c); but application of isotonic CaCl_2 restores normal responses within a minute (d). The spontaneous rhythms are similarly suppressed by potassium excess and restored by calcium. Potassium may also reverse the polarity of the waves (Fig. 3Be).

Calcium ion. Intravascular injection of isotonic CaCl_2 , sufficient to increase the blood concentration 50 to 200 per cent, depresses the animal, if under light anesthesia. Within 2 min. the fast irregular geniculate potentials disappear, the 2 per sec. rhythm is regularized and enhanced, and the amplitude and duration of the after-discharge to light is decreased (Fig. 3Ad and e). The increased and stabilized slow rhythm now persists during optic stimulation, which normally disrupts it. Intravascular Ca ion excess was found to be less toxic than K ion excess, and calcium action could be tested within 20 min. of a preceding potassium injection.

Local application to the optic cortex of Ringer's solution with three times the normal calcium does not alter potentials, but isotonic CaCl_2 depresses evoked and spontaneous ones during 10 min. (Fig. 3Bf and g). The calcium depression is antagonized by potassium or by citrate, which promptly restores normal responses. Calcium ion deficiency, produced by intravascular injection of 2.0 cc. of 3.0 per cent Na citrate in Ringer's solution, acts upon lateral geniculate potentials much like potassium excess.

Hydrogen ion. Intravascular injection during 30 sec., of 1.0 cc. per kg. of 0.01 N HCl in Ringer's solution increases respiratory depth and evokes general restlessness and movement. It also disrupts the slow lateral geniculate rhythm, decreases the amplitude of spontaneous and evoked potentials, and prolongs the after-discharge. The rhythm does not reappear for 20 to 30 min. Local application of approximately 0.001 N HCl in Ringer's solution (unbuffered) evokes, within 2 min., wave trains at 7 to 12 per sec. and 40 to 60 μ V. These reappear during retinal stimulation after the initial effect has worn off.

An increase of pH is more toxic than a decrease, but less prolonged in action. Rapid injection of 2 cc. of 0.01 N acid or 1.5 cc. of 0.001 N alkali per kg. is usually fatal. (Barbitalized dogs can tolerate as much as 3 cc. of N HCl injected slowly during 3 min., Harkins and Hastings, 1931; the maximal pH fall, to 6.92, occurs in 6 min.) Smaller amounts of NaOH depress respiration and general motor activity, immediately disrupt the slow geniculate rhythm, and markedly diminish the spontaneous and evoked poten-

tials. Normal potentials return in 6 to 8 min. At these pH extremes, acid and alkali injections do not consistently counteract one another.

Lactate ion. Less than 2 min. after the intravascular injection of 2.0 cc. of 4 per cent Na lactate, the slow geniculate rhythm is less regular and smaller and is largely replaced by rapid, large, irregular potentials. These are especially prominent in the after-discharge following optic stimulation. The modified potentials persist for 1 to 2 hr.

Strychnine. Within 1 min. after the intravascular injection of 2.0 cc. of 1.0 per cent strychnine solution, the slow geniculate rhythm is augmented; often, when no obvious rhythm is present in a dark-adapted cat, strychnine initiates conspicuous rhythmic potentials. During optic stimulation the augmented slow rhythm is replaced by waves at 13 per sec. and 15 μ V., to reappear almost immediately after the "off" (Fig. 3Af and g). When some minutes have elapsed following the strychnine injection, the rapid rhythm appears during illumination and continues as an after-discharge. The high frequency potentials of potassium excess, in contrast, appear primarily during light and less frequently in the after-discharge.

Blood sugar. Nembutal anesthesia causes no change in mean blood sugar level (Hrubetz and Blackberg, 1938). Insulin, in a dose of 10 to 15 units per kg. subcutaneously, does not lead to gross convulsions, though geniculate potential changes are definite within an hour. (Compare the time course of blood sugar changes; Zucker and Berg, 1937. Smaller insulin injections do not affect potentials.) The background potentials become augmented and the normal diphasic "on" and "off" responses to light are replaced by 3 to 5 large oscillations, mainly positive, followed by a prolonged high-frequency after-discharge (Fig. 4A). The picture is similar to that induced by potassium excess. Similar quantities of insulin, given intravenously, lead to repetitive optic responses, but they markedly decrease the spontaneous potentials (Fig. 4B). In both cases, the altered responses to light remain constant with repeated stimuli. The normal picture is restored within 5 min. by intravascular glucose (2 cc. of a 40 per cent solution). Hyperglycaemia leads after some 10 min. to an increase in rate and amplitude of the slow rhythm (Fig. 4Ag and h).

Hypoxia. Bilateral occlusion, by traction, of the carotid arteries reversibly abolishes the spontaneous and evoked potentials in the lateral geniculate body. Electrical activity disappears usually in about 1 min., and returns in 1 to 3 min. after release. Optic responses are lost at the same time as the spontaneous ones. In 3 animals with transected brain stem (and loss of vertebral blood) the average times of loss and return of potential were 50 sec. and 80 sec., as compared with 240 and 400 sec. in 3 intact animals. Before anoxic failure and early during subsequent recovery, potentials are increased, with a large high-frequency component.

DISCUSSION

The unevoked potential rhythms of central neurones are controlled by their own states of metabolism, membrane charge, etc., which in turn are

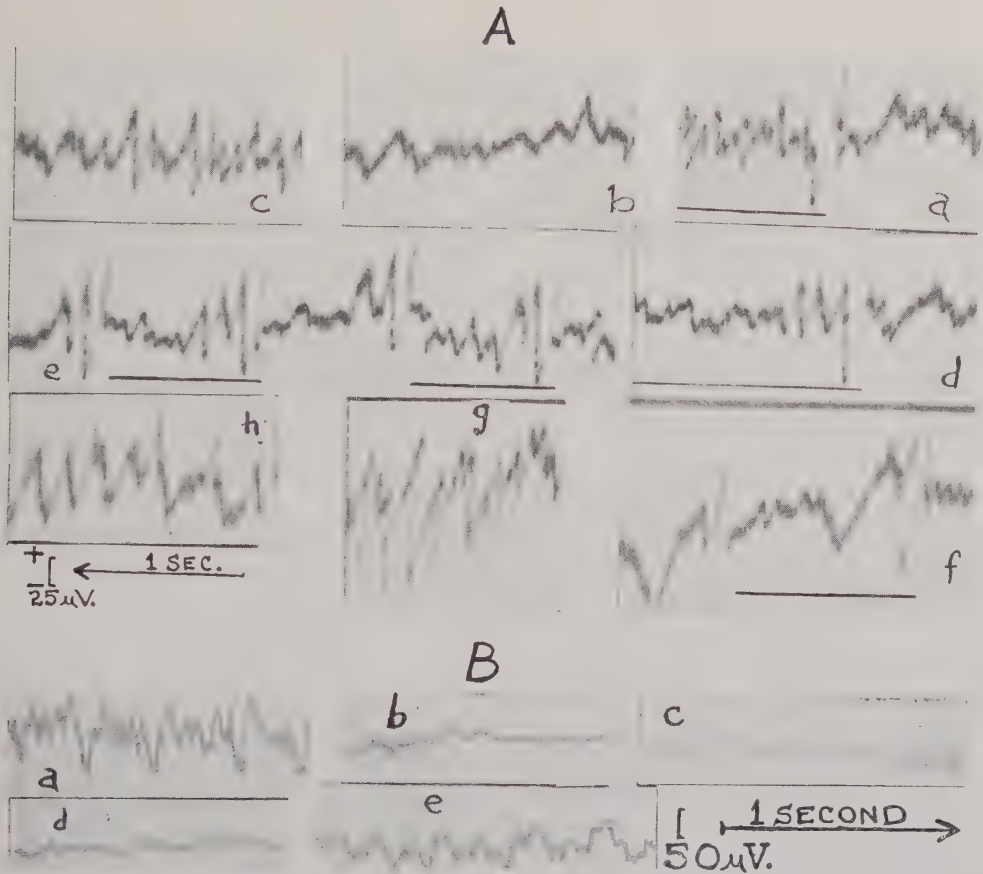


FIG. 4A. Lateral geniculate potentials as altered by change in blood sugar: (a) Normal "on"; (b) 2 sec. and (c) 10 sec. after "off"; (d) 20 min. after insulin (10 units subcut.) single "on" and (e) repeated optic stimuli, note multiple waves; (f) 6 min. after (e) and intravenous glucose, "on" and "off"; (g) 2 and (h) 20 sec. after (f).

FIG. 4B. Like Figure 4A, undulator ink records obtained at Ohio State University. (a) Before insulin; (b) 70 min. after intravenous insulin, dark, (c) during light, (d) dark, 2 min. later; (e) 8 min. later, following intravenous glucose.

modified by influent nerve impulses and the condition of the surrounding medium. As recorded, these potentials involve the further factors of the number, kind, and distribution of active cells or cell groups and the degree of unison of their separate beats. Interpretation of such records at present remains rather circumstantial for the mammalian brain. Cells of the lateral geniculate nucleus are roughly stratified with the largest ones located most ventrally. Of the four more or less distinct rhythms, the faster are more prominent in the ventral portion. Perhaps the larger cells normally manifest a faster rhythm, but at all needle positions there is considerable play of potential rate from time to time under seemingly fixed conditions.

The slow 3 per sec. rhythm, seen especially in the geniculate, might arise in the eye and be normally imposed on other portions of the optic system.

It is seen in the primary tract and, after abolition by light, reappears in it earlier than in the geniculate. On the other hand, the geniculate rhythm persists for some hours after severing the optic nerves, so that the thalamic cells can maintain the beat alone. It similarly survives, is even enhanced by, the removal of the optic cortex, so that any thalamo-cortical cycle which may exist is not essential. The same is true for connections with the mesencephalon, section of them favors the diencephalic rhythm (see also Bremer, 1935), and even full synaptic blockade with nicotine leaves the geniculate active for a time (Libet and Gerard, 1938). These neurones, then, do not depend on immediate nerve impulses from eye, cortex, or brain stem to maintain their beat.

Over longer periods this may not be true; also, at any given moment, the local behavior is modifiable by incoming impulses. The absence of the rhythm after prolonged dark and its return in moderate diffuse light can be interpreted in terms of a state of cell excitation which slowly falls below threshold for the rhythm when no afferent impulses are being received. Any enhancement of the general level of cell excitation (whatever the metabolic, membrane, or other mechanism for controlling this level may be) favors the potential waves; and they are increased during diffuse and after strong optic stimulation and by potassium and strychnine.

The disruption of the rhythm during strong light has been generally interpreted as a desynchronization of the many cells, rather than as a decrease in the beat of each. The effect of polarization is presumably just the reverse of this. A rhythm, initially feeble and more or less obscured by the partial asynchrony of cells, is greatly increased and regularized (but not altered in form or rate), as an imposed potential sweeps the cells into unison. The change is independent of the direction of the imposed current, which in any event flows across irregularly oriented cell processes, and outlasts the current for many minutes. The current may, of course, alter the beat of individual cells as well as their phase relations. On this there is no evidence except that a cell stimulation would probably increase rate, which occurred in only one case.

The influence of potassium increase and calcium increase or decrease (citrate), on optic potentials is in accord with their action on neurone metabolism and activity (see Gerard, 1938). Moderate increase of potassium enhances irritability and excitation, and it augments potentials, especially the faster rhythms. Greater concentrations of potassium, applied locally to the cortex, depress irritability and potentials. Calcium decrease acts similarly, excess oppositely, and increase of both potassium and calcium antagonizes the action of excess of either alone. After-discharge following optic stimulation is prolonged by potassium, shortened by calcium. (Cf. motor discharge after cerebellar stimulation; Gerard and Magoun, 1937.) The dominant 3 per sec. wave of the geniculate is not abolished by calcium injections but, due to suppression of faster waves, remains more clear than normal. It may be actually strengthened, or at least cell synchrony made more positive, by increased calcium since light no longer disrupts it. Possibly

cells, which at "normal" excitation levels produce the faster rhythms, fall into the slower tempo when somewhat depressed by calcium.

The marked increase and decrease of pH studied here both disrupt the slow rhythm. Acidity increases the 8 per sec. waves and prolongs after-discharge, alkalinity depresses all waves, including evoked potentials and their after-discharge. Over a finer range, cortical potentials are increased by an alkaline shift in the cortex and diminished by an acid one (Dusser de Barenne, McCulloch, and Nims, 1937), in parallel to changes in irritability. The action of strychnine (by injection), in initiating or increasing a regular slow rhythm and in making more rapid and intense the after-discharge following physiological stimulation, requires no comment. Strychnine spikes were not seen in any of these experiments as on local application. (See Jasper, 1937; Dusser de Barenne and McCulloch, 1938.)

Of especial interest are the effects of hypoglycaemia (insulin) and of glucose injection. Others have reported, for the cortex, mainly of human subjects, a slowing of the normal rhythms under insulin action (Hoagland, Rubin and Cameron, 1937; Lennox, Gibbs, and Gibbs, 1938). The "delta index," of slow wave components, inversely follows blood sugar concentration, sometimes quite accurately. Such slowing has been interpreted as evidence of a decreased metabolism of the active neurones. On the other hand, hypoglycaemia acts much like hypoxia, which induces increased neural sensitivity and discharge as a transient phase between the normal and depressed states. (Gerard, 1938.) The present findings for the geniculate indicate such an excitation. Under insulin action (and promptly abolished by glucose) the spontaneous rhythm is enhanced, the "on" and "off" light responses are larger than and show complex potential swings not present in the normal ones, and the after-discharge is more prolonged and of a higher frequency. Hypoglycaemia, in fact, affects geniculate potentials much as does increased blood potassium—even to a reversal of the excitant action when concentration changes are excessive. Insulin causes a decrease in blood potassium (Keyes, 1938) while anoxia causes an increase (Mullin, Dennis and Calvin, 1938). In the latter case the potassium content of the brain is, if anything, increased (Gerard and Tupikova, unpublished), the potassium entering into circulation from other sources. Under insulin, also, it is possible that brain potassium is increased, some entering from the blood. This has yet to be determined. By whatever means low blood or brain carbohydrate produces its action, it does seem that it is able to increase brain activity as well as to depress it.

SUMMARY

The normal spontaneous rhythms of the cat's geniculate body, especially the dominant one at 3 per sec., are independent of impulses reaching these neurones from optic nerves, cortex or brain stem. The background level of excitation, determined largely by optic impulses, however, strongly influences their character. The slow rhythm fades out over hours in the dark and is reinitiated after brief illumination.

The enhanced spontaneous and evoked optic potentials induced by potassium, citrate, acid, strychnine, insulin and polarizing currents, and the diminished potentials resulting from calcium, alkali and glucose are described and interpreted.

REFERENCES

- BARTLEY, S. H. Relation of intensity and duration of brief retinal stimulation by light to electrical responses of optic cortex of rabbit. *Amer. J. Physiol.*, 1934, 108: 397-408.
- BARTLEY, S. H. Temporal and spatial summation of extrinsic impulses with intrinsic activity of cortex. *J. cell. comp. Physiol.*, 1936, 8: 41-62.
- BISHOP, G. H. Cyclic changes in excitability of optic pathway of rabbit. *Amer. J. Physiol.*, 1935, 103: 213-224.
- BISHOP, G. H. Interpretation of cortical potentials. *Cold Spr. Harb. Symp. quant. Biol.*, 1936, 4: 305-317.
- BREMER, F. Cerveau "isolé" et physiologie du sommeil. *C. R. Soc. Biol., Paris*, 1935, 118: 1235-1241.
- DUBNER, H. Further studies of factors influencing brain rhythms. *Amer. J. Physiol.*, 1938, 123: 56-57.
- DUSSER DE BARENNE, J. G., and McCULLOCH, W. S. Functional organization in the sensory cortex of the monkey (*Macaca mulatta*). *J. Neurophysiol.*, 1938, 1: 69-85.
- DUSSER DE BARENNE, J. G., McCULLOCH, W. S., and NIMS, L. F. Functional activity and pH of the cerebral cortex. *J. cell. comp. Physiol.*, 1937, 10: 277-289.
- GERARD, R. W. Factors controlling brain potentials. *Cold Spr. Harb. Symp. quant. Biol.*, 1936, 4: 292-298.
- GERARD, R. W. Anoxia and neural metabolism. *Arch. Neurol. Psychiat., Chicago*, 1938, 40: 985-996.
- GERARD, R. W., and DUBNER, H. Factors influencing brain potentials. *Trans. Amer. neurol. Ass.*, 1936, 62: 55-60.
- GERARD, R. W., and MAGOUN, H. W. Influence of potassium and calcium on motor discharges. *Proc. Soc. exp. Biol., N. Y.*, 1936, 34: 755-756.
- GERARD, R. W., MARSHALL, W. H., and SAUL, L. J. Electrical activity of the cat's brain. *Arch. Neurol. Psychiat., Chicago*, 1936, 36: 675-738.
- HARKINS, H. N., and HASTINGS, A. B. A study of electrolyte equilibrium in the blood in experimental acidosis. *J. biol. Chem.*, 1931, 90: 565-595.
- HOAGLAND, H., RUBIN, M. A., and CAMERON, D. E. Electroencephalogram during insulin hypoglycemia. *Amer. J. Physiol.*, 1937, 120: 559-570.
- HOUSSAY, B. A., and MARENZI, A. D. Fixation of potassium injected intravenously. *Rev. Soc. argent. Biol.*, 1937, 13: 139-151.
- HRUBETZ, M. C., and BLACKBERG, S. N. The influence of nembutal, pentothal, seconal, amytal, phenobarbital, and chloroform on blood sugar concentration and carbohydrate mobilization. *Amer. J. Physiol.*, 1938, 122: 759-764.
- JASPER, H. H. Electrical signs of cortical activity. *Psychol. Bull.*, 1937, 34: 411-481.
- KEYES, A. The effects in man and dogs of massive doses of insulin on the composition of the blood serum. *Amer. J. Physiol.*, 1938, 123: 608-613.
- LENNOX, W. G., GIBBS, F. A. and GIBBS, E. L. The relationship in man of cerebral activity to blood flow and to blood constituents. *Res. Publ. Ass. nerv. ment. Dis.*, 1938, 18: 277-297.
- LIBET, B. and GERARD, R. W. Automaticity of central neurones after nicotine block of synapses. *Proc. Soc. exp. Biol., N. Y.*, 1938, 38: 886-888.
- LORENTE DE NÓ, R. Studies on the structure of the cerebral cortex. II. Continuation of the study of the ammonic system. *J. Psychol. Neurol., Lpz.*, 1934, 46: 113-177.
- LORENTE DE NÓ, R. Facilitation of motoneurones. *Amer. J. Physiol.*, 1935, 113: 505-523.
- MULLIN, F. J., DENNIS, J., and CALVIN, D. B. Blood potassium in tetany and asphyxia. *Amer. J. Physiol.*, 1938, 124: 192-201.
- TUPIKOVA, N., and GERARD, R. W. Salt content of neural structures. *Amer. J. Physiol.*, 1937, 119: 414-415.
- WANG, G. H. Action potentials of visual cortex and superior colliculus induced by stimulation of retina with light. *Chinese J. Physiol.*, 1934, 8: 121-141.
- ZUCKER, T. F., and BERG, B. N. Course of blood sugar after intravenous insulin in normal dogs and cats. *Amer. J. Physiol.*, 1937, 119: 531-538.

